

# THE ASSOCIATION BETWEEN OBESITY AND PERIODONTAL DISEASE

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# THE ASSOCIATION BETWEEN OBESITY AND PERIODONTAL DISEASE

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The *American Journal of Public Health* (2004) editorializes obesity as '*the public health challenge of our time.*' Obesity creates risk for chronic health problems, is associated with increased mortality and exists in complexes of multiple, clustered behavioral risk factors. *Fine* (2004) identifies obesity among the four most common risk factors of chronic disease [*cigarette smoking, risky drinking of alcohol, physical inactivity, overweight*]. That set of risk factors applied to the 2001 National Health Interview Survey showed 17 percent (among 29,000) possessed three or more. Obesity with raised cholesterol and hypertension is the major cause of mortality and disease in Europe (*James 2004*) and is an issue for developing countries as well (*Caballero 2001*).

*Adipocytes* in the adipose (fatty) tissues of obese people produce quantities of active molecules like leptin, important in regulating energy expenditure (*thus body weight*). Adipocyte-derived active molecules (*adipocytokines*) are candidates for the close association between obesity and multiple risk factor syndromes.

Similarly periodontal disease is one of the world's most common chronic diseases [possibly 35 percent of U.S. adults ages 30-40]. Increasing evidence establishes periodontal disease as a significant risk factor in the etiology of diseases with inflammatory components. [Severe periodontal disease is the well-established sixth complication of diabetes (L  e 1993) and the relationship may be two-way (Grossi

1998).] Obesity is a systemic disease predisposing to co-morbidities and complications that affect overall health; cross-sectional research suggests obesity is associated with periodontal diseases by underlying biologic mechanisms yet to be established. Linking evidence in the pathophysiology of both points toward inflammatory processes: The proinflammatory cytokine *tumor necrosis factor-alpha (TNF-alpha)* is produced by adipocytes and the concentration of TNF-alpha is elevated in the obese, declining with weight loss. TNF-alpha suppresses insulin action via its specific receptor. Hence TNF-alpha exacerbates insulin resistance.

The purpose of this research is to determine if subjects with obesity have a higher chance of developing periodontal disease, using statistical analyses of a retrospective review of the University of Pittsburgh School of Dental Medicine electronic health record [EHR].

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## **PREFACE**

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Lastly the author dedicates this work to his parents for their encouragement, and to Dana.

## 1.0 INTRODUCTION

Obesity creates risk for chronic health problems, is associated with increased mortality and exists in complexes of multiple, clustered behavioral risk factors. Similarly periodontal disease is one of the world's most common chronic diseases [possibly 35 percent of U.S. adults ages 30-40]. Increasing evidence establishes periodontal disease as a significant risk factor in the etiology of diseases with inflammatory components. Severe periodontal disease is the well-established sixth complication of diabetes (Løe 1993) and the relationship may be two-way (Grossi 1998). Obesity is a systemic disease predisposing to co-morbidities and complications that affect overall health; cross-sectional research suggests obesity is associated with periodontal diseases by underlying biologic mechanisms yet to be established. Linking evidence in the pathophysiology of both points toward inflammatory processes: The proinflammatory cytokine *tumor necrosis factor-alpha (TNF-alpha)* is produced by adipocytes and the concentration of TNF-alpha is elevated in the obese, declining with weight loss. TNF-alpha suppresses insulin action via its specific receptor. Hence TNF-alpha exacerbates insulin resistance.

This research is a retrospective review of the University of Pittsburgh School of Dental Medicine electronic health record [EHR].

## **1.1 REVIEW OF THE LITERATURE: PERIODONTAL DISEASE**

### **1.1.1 The Epidemiology of Periodontal Disease**

Epidemiology is the study of health and disease in populations and of how these states are influenced by heredity, biology, physical environment, social environment, and personal behavior. In the 1940s, early epidemiologic studies of periodontal disease started with indices of gingivitis. By the 1980s the research focused more on the extent and severity of the disease (Reddy and Jeffcoat 1993). Thirty-six percent (36%) of the population in the United States aged 19 years and older was found to be affected with periodontitis (pockets greater than 4mm) when Brown et al. in 1989 reported on a national survey done by the Triangle Institute in 1981. Findings also included that 8% of the population was affected with advanced periodontitis (pockets greater than 6mm), and that 4% of the population required extraction of teeth due to end stage periodontal destruction. Between 1985-1986, the NIDCR conducted a national survey of employed adults. Of adults over 35 years of age, 18% showed attachment loss of greater than 4 mm (Brown et al. 1990). That same national survey also showed that 47% of males and 39% of females exhibited at least one site that bled on probing. Findings from the National Center for Health Statistics surveys in the 1960s, 1970s, and the 1985 and 1986 NIDR adult survey were compared by Capilouto and Douglass (1988). About 20% of adults in the United States showed a level of periodontal disease severity that required professional treatment. Multiple investigators recently have documented that 5% to 15% of any population exhibits severe generalized periodontitis (NIDR 1987,

CDC 1997, Oliver et al. 1998). Similar results have been shown when international epidemiologic studies were conducted on the prevalence of periodontal disease.

### **1.1.2. Clinical Assessment of Periodontal Disease**

Apart from gingival bleeding and radiographic assessment of bone loss, clinical attachment loss (CAL) and probing depth (PD) have historically been the basic clinical measures for periodontitis utilizing the manual probe, a protocol that was described more than 45 years ago (Ramfjord 1959).

However, in an effort to quantify etiologic agents, gingival alterations or alterations in the attachment apparatus, many periodontal indices have been used and described. Such indices have been used to measure symptoms of disease, the etiologic factors involved, to measure the disease, and also in the treatment of diseases. According to Barnes et al. (1986), indices may be used in epidemiologic surveys, clinical trials, or to evaluate the progress of an individual patient and patient motivation.

The PMA Index published by Schour and Massler (1947) was the first system to classify gingivitis. The PMA Index determined the number of (1) inflamed papillae (P), (2) gingival margins (M), and (3) areas of attached gingiva (A) at the labial surfaces of anterior teeth. The PMA Index helped to examine and compare prevalence and severity scores from different populations, considering its limitations (Lindhe 2008).

The Periodontal Index (PI) developed by Russel in 1956 examined both gingivitis and periodontitis. The Periodontal Index was deemed useful for epidemiologic surveys but not clinical trials due to its lack of sensitivity to small differences (Russel 1956).

Qualitative changes in the gingival soft tissue were measured by the gingival index developed by Löe and Silness in 1963. Scores from 0-3 on the four smooth surfaces of each tooth were used, with (0) being normal, and (3) describing severe inflammation (Löe 1967).

More indices were developed in an effort to determine treatment needs. Some of these indices include the Periodontal Treatment Need System (PTNS) developed by Johansen et al. (1973). This index provided a quick measurement of treatment needs without attention to details (Lindhe 2008). Another index used in epidemiologic studies was the Community Periodontal Index of Treatment Needs (CPITN). It was developed by Ainamo in 1982, and used a special World Health Organization (WHO) periodontal probe to assess treatment needs (Ainamo et al. 1982).

Lastly is an index developed based on the CPITN, known as the Periodontal Screening and Recording (PSR). Similar to the CPITN, the PSR index divides the dentition into six sextants, and uses a color coded probe. The PSR examines all the teeth but only records on a scale from 0-4 the worst score, and based on the severity of the score (4 being most severe) the periodontal management and treatment are determined (Lindhe 2008).

### **1.1.3. The Etiology of Periodontal Disease**

The presence of bacterial plaque is required for the development of gingivitis (Löe et al. 1965, Theilade et al. 1966). It is thought that bacterial plaque will induce pathological changes in the tissue directly and indirectly (Page 1986). The mass of that bacterial plaque rather than specific periodontal pathogens causing periodontitis explains the

nonspecific plaque theory. More recently, research has shown that specific bacteria are responsible for periodontitis, and that describes the specific plaque theory used today (Jeffcoat 1993). Smalley in 1994 further explains the previous (nonspecific plaque) theory suggesting that organisms display virulence factors giving them the ability to mediate tissue destruction directly or indirectly.

In the periodontal pocket, more than 300 species of microorganisms have been isolated, with only a small percentage being etiologic agents (Genco et al. 1990, Moore et al. 1994). Bacterial constituents or metabolites capable of either causing disruption of homeostatic or protective host mechanisms or causing the progression or initiation of a disease are termed bacterial virulence factors. Such factors are one of the characteristics to imply or deem an organism as etiologic (The American Academy of Periodontology 1999). For periodontal microorganisms to act as pathogens, the organism must exhibit 1) the capacity to colonize; 2) the ability to evade antibacterial host defense mechanisms; and 3) the ability to produce substances that can directly initiate tissue destruction (The American Academy of Periodontology 1999).

In 1992, Socransky and Haffajee concluded that for disease to result from a pathogen, the potentially-pathogenic organism 1) must be a virulent clonal type; 2) must possess the chromosomal and extra-chromosomal genetic factors to initiate disease; 3) must discover a host susceptible to this particular pathogen; 4) must be in numbers sufficient to exceed the threshold for that host; 5) must be located at the right place; 6) must be fostered by other bacterial species, or at the very least not inhibited by other bacteria, during the development of the pathologic process; and 7) must establish itself in a local environment conducive to the expression the species' virulence properties.

A dynamic equilibrium exists between periodontal disease and health, and the equilibrium and its disruption may be influenced by many factors including local and systemic (Smalley 1994).

#### **1.1.4.Risk Factors for Periodontal Disease**

A risk factor is an environmental exposure, aspect of behavior, or an inherent characteristic associated with a disease (Last 2001).

Risk factors for periodontal disease include oral hygiene, education, systemic factors, socioeconomic status, and age (Reddy and Jeffcoat 1993).

Systemic factors and modulation of host response will be discussed in detail in later sections. An example of such systemic modulation of host response would be how diabetes mellitus alters the host defense mechanisms and decreases the ability of the host-body to fight infection (Reddy and Jeffcoat 1993). Further evidence for the systemic modulation of host response in the case of risk factors for periodontal disease is the conclusion by Grossi et al. (1994) that diabetic subjects experience twice more odds for more attachment loss than non-diabetics.

An association between cigarette smoking and periodontitis has been shown in both cross-sectional and longitudinal studies (Ismail and Lewis 1993). A history of smoking is highly correlated with severity of attachment loss (Gross et al 1994) when a cross sectional study of risk indicators for attachment loss was conducted.

Multiple investigators have shown that age was associated with severity of attachment loss (Hugoson et al. 1992, Grossi et al. 1994). However other studies have concluded that it is uncommon for elderly people with reasonably intact dentition to

exhibit sudden bursts of periodontitis (Page 1984). And when exhibited by little gingival inflammation, and few deep pockets, tooth retention, good oral hygiene, and periodontal health are closely associated, regardless of age (Burt et al. 1985, Abdellatif et al. 1987).

Lower socioeconomic status (SES) has been associated with periodontal disease as a risk factor (National Center for Health Statistics 1965, 1979). Generally people who have a higher SES tend to practice better oral hygiene, have more positive attitudes toward oral hygiene, and visit the dentist more frequently.

## **1.2 REVIEW OF THE LITERATURE: DIABETES MELLITUS**

*Diabetes mellitus* is a chronic metabolic disorder characterized by the inability to maintain blood glucose levels within normal physiological limits. Diabetes mellitus results from the inadequate production of insulin or insulin resistance, both creating a physiologic condition in which the body cannot respond to the insulin formed within the system leading to what is called hyperglycemia, the name of the condition deriving from the state of accumulation of glucose in the blood. Insulin is a hormone produced by the Islets of Langerhans in the pancreas, and is responsible for the absorption of glucose into the cells for their energy needs, and into the liver and fat cells for storage.

Diabetes mellitus has been recognized as a serious public health problem resulting in significant morbidity and mortality due to specific diabetes-related microvascular complications, increased risk of macrovascular complications (ischemic heart disease, stroke and peripheral vascular disease), and increased inflammatory mediator response (American Diabetes Association 2005).



## **1.2.1 Types of Diabetes Mellitus**

**1.2.1.1 Prediabetes** Prediabetes describes a condition in which blood glucose levels are higher than normal but not high enough to be labeled diabetes. Prediabetes occurs when the fasting blood sugar is between 101mg/dL and 126mg/dL or if the blood sugar level two hours after glucose tolerance test is between 140mg/dL and 200mg/dL (American Diabetes Association 2005). It has been shown that people with prediabetes have an increased risk for future Type II diabetes as well as heart disease (American Diabetes Association 2005). Borderline diabetes, Impaired Glucose Tolerance (IGT), Impaired Fasting Glucose (IFG), Impaired Glucose Regulation (IGR), and Non-Diabetic Hyperglycemia (NDH) are also medical terms that used to describe a prediabetes condition (American Diabetes Association, 2005).

**1.2.1.2 Type I Diabetes Mellitus** Type I diabetes mellitus was formerly called insulin-dependent diabetes or juvenile-onset diabetes. In this type, more than 90 percent of the pancreatic Beta cells are permanently destroyed due to cellular mediated auto-immune destruction, leading to insulin deficiency (Mealey and Oates 2006). The use of exogenous insulin is a must to compensate the lack of insulin production and to sustain life, as those patients may develop diabetic ketoacidosis, a life-threatening condition, hence the former name “insulin-dependent diabetes” (Mealey and Oates 2006). Although some studies showed 15-30 percent of all patients being diagnosed

after the age of 30, Type I diabetes usually occurs before that age, in children and adolescents (Mealey and Oates 2006).

**1.2.1.3 Type II Diabetes Mellitus** Type II diabetes mellitus is also called non-insulin-dependent diabetes or adult-onset diabetes. It is the most common type of diabetes, which has a gradual onset and is often without symptoms; usually found in people over 30, Type II diabetes mellitus becomes more common with the aging process. However, in the presence of escalating numbers of overweight young people and growing rates of obesity status, Type II diabetes mellitus increasingly occurs at younger ages (American Diabetes Association 2005). Type II diabetes develops when the body does not produce enough insulin. Type II diabetes also occurs in situations where sometimes insulin is produced even at higher than normal levels but does not work properly or as well as it should, so that there is not enough insulin to meet the body needs. This condition is insulin resistance. Type II diabetes can be controlled by lifestyle changes, careful dietary management, regular physical activity, weight reduction, and oral anti-diabetic medications, but in some instances, insulin is required ([www.diabetes.org.uk/guide-to-diabetes](http://www.diabetes.org.uk/guide-to-diabetes)).

## **1.2.2 Symptoms of Diabetes Mellitus**

Both types of diabetes have very similar symptoms related to the direct effect of high blood glucose levels. The body tries to flush the excess glucose in its system out in the

urine, resulting in these classic symptoms: polyuria (passing urine frequently), polydipsia (increased thirst), polyphagia (increased appetite), blurred vision, extreme fatigue, delayed wound healing, genital itching, weight loss, and regular episodes of thrush ([www.diabetes.org.uk/guide-to-diabetes](http://www.diabetes.org.uk/guide-to-diabetes)).

In Type I diabetes, signs and symptoms are usually obvious and develop rapidly (over few weeks). Once the condition is treated and under control, these symptoms are quickly relieved. On the other hand, symptoms in Type II diabetes are not as obvious, as the blood glucose level may rise so slowly that patients may not have symptoms for years before they are diagnosed. Often Type II diabetes is diagnosed during an appointment for another health problem or during a routine medical checkup ([diabetes.org.uk/guide-to-diabetes](http://diabetes.org.uk/guide-to-diabetes)).

### **1.2.3.Diagnosis of Diabetes Mellitus**

In 1965, the World Health Organization (WHO) published diagnostic criteria for diabetes mellitus. The last review of these criteria (1998) was published as the guidelines for the *Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications* (American Diabetes Association 2005). Since then, more information regarding the diagnosis has become available. In 2003, the American Diabetes Association (ADA) reviewed its diagnostic criteria, and the last review was in 2005. The following Table shows the current diagnostic parameters for diabetes mellitus.

**TABLE 1.**

**2005 American Diabetes Association Criteria for the Diagnosis of Diabetes Mellitus, Impaired Glucose Tolerance (IGT), and Impaired Fasting Glucose (IFG)**

	Normal	Diabetes	IGT	IFG
Fasting plasma glucose (mg/dl)	<100	≥126		100-125
Casual plasma glucose (mg/dl)		≥200 plus symptoms of diabetes		
2-hour PG (mg/dl)**	<140	≥200	140-199	

\*Table 1. is adopted from Mealey BL. Oates TW. Diabetes mellitus and periodontal diseases. *J Periodontol* 2006;77(8):1289-1303.

\*\*2-hour postload glucose (PG) using the 2-hour oral glucose tolerance test

Currently, medical scientists utilize three methods to diagnose diabetes, and any positive laboratory value should be confirmed on a different day, because of the fact that a single abnormal laboratory result is not sufficient for definitive diagnosis (Mealey and Oates 2006). These three methods are:

- 1) The classic symptoms of diabetes (polyuria, polydipsia, and unexplained weight loss) with casual plasma glucose concentration  $\geq 200$  mg/dl ( $\geq 11.1$  mmol/l). *Casual* is defined as any time of day without regard to the time since the last meal (Mealey and Oates 2006).
- 2) Fasting plasma glucose (no caloric intake at least eight hours) is  $\geq 126$  mg/dl ( $\geq 7.00$  mmol/l); and

- 3) Two-hour post-load glucose  $\geq 200\text{mg/dl}$  ( $\geq 11.1\text{mmol/l}$ ) during the oral glucose tolerance test (Mealey and Oates 2006).

In order to monitor the patient's overall glycemic control (after being diagnosed with diabetes), the hemoglobin A1c (HbA1c) test is used. Research has concluded that to date no gold standard assay exists for the HbA1c and many countries do not have ready access to the test. For those reasons, the HbA1c test is not recommended for diagnosis of diabetes mellitus (Mealey and Oates 2006).

However the HbA1c test can be used to determine glycohemoglobin levels and gives an estimate of the average blood glucose level over 30-90 days preceding the test (Mealey and Oates 2006). The HbA1c 'normal' is less than 6% [as shown in Table 2].

Investigators have observed that HbA1c levels correlate well with the appearance of diabetic complications as seen in Table 3, and it may become a promising test for the diagnosis of diabetes in the future (Mealey and Oates 2006).

**TABLE 2.**  
**American Diabetes Association Recommendations for HbA1c Levels**

<b>HbA1c%</b>	<b>Interpretation</b>
<b>&lt;6</b>	Normal value
<b>&lt;7</b>	Treatment goal for the diabetic patient Dietary management, physical activity, and/or medications should control glucose level properly to maintain this value
<b>&gt;8</b>	Medical intervention by physicians (diabetologists) to manage the condition is recommended to improve glycemic control

\*Table 2. is adopted from Mealey BL. Oates TW. Diabetes mellitus and periodontal diseases. *J Periodontol* 2006;77(8):1289-1303.

**TABLE 3.**  
**Correlation between HbA1c and Mean Plasma Glucose Levels**

<b>HbA1c%</b>	<b>Mean Plasma Glucose (mg/dl)</b>
<b>6</b>	<b>135</b>
<b>7</b>	<b>170</b>
<b>8</b>	<b>205</b>
<b>9</b>	<b>240</b>
<b>10</b>	<b>275</b>
<b>11</b>	<b>310</b>
<b>12</b>	<b>345</b>

\*Table 3. is adopted from Mealey BL. Oates TW. Diabetes mellitus and periodontal diseases. *J Periodontol* 2006;77(8):1289-1303.

#### **1.2.4.Epidemiology and Prevalence of Diabetes Mellitus**

The prevalence of diabetes for all ages is demonstrably increasing globally due to population growth, aging, urbanization, and high obesity prevalence and physical inactivity. This growing epidemic will lead to increased morbidity and mortality and greater expenditure of healthcare dollars (Jeffrey Freeman 2010).

In the United States, prevalence and incidence of diabetes is growing annually and varies by age and racial category (National Center for Health Statistics U.S. 2005). Approximately 21 million Americans were affected including more than nine percent of the U.S. adult population, including six million of these individuals diabetic but undiagnosed. In 2002, almost 1.3 million new patients were diagnosed with diabetes; an increase of 500,000 new cases every year since 1998. Most of the diabetic cases (about 85-90%) were Type II diabetes, whereas only 5-10% were Type I diabetes. The

increase in prevalence and incidence of both types of diabetes was mainly because of dramatic increases in the rate of obesity among Americans (National Center for Health Statistics U.S. 2005).

Wild and co-workers have investigated the global prevalence of diabetes for all age groups in their study for the year 2000, with projections for 2030 (Wild et al, 2004). The global prevalence of diabetes for all age groups was estimated to be 2.8% in 2000, increased to 4.4% in 2030, with the total number of adult diabetics (aged  $\geq 20$  years) worldwide projected to increase from 171 million in 2000 to 366 million in 2030 (Wild et al., 2004). The highest number of diabetic cases has been found in India, followed by China, and then the United States, as seen in Table 4, below.

**TABLE 4.**  
**Countries with the Highest Estimated Prevalence of Diabetes 2000 vs. 2030**

<b><u>2000</u></b>			<b><u>2030</u></b>	
<b>Rank</b>	<b>Country</b>	<b>Prevalence (millions)</b>	<b>Country</b>	<b>Prevalence</b>
<b>1</b>	<b>India</b>	<b>31.7</b>	<b>India</b>	<b>79.4</b>
<b>2</b>	<b>China</b>	<b>20.8</b>	<b>China</b>	<b>42.3</b>
<b>3</b>	<b>United States</b>	<b>17.7</b>	<b>United States</b>	<b>30.3</b>
<b>4</b>	<b>Indonesia</b>	<b>8.4</b>	<b>Indonesia</b>	<b>21.3</b>
<b>5</b>	<b>Japan</b>	<b>6.8</b>	<b>Pakistan</b>	<b>13.9</b>
<b>6</b>	<b>Pakistan</b>	<b>5.2</b>	<b>Brazil</b>	<b>11.3</b>
<b>7</b>	<b>Russian Federation</b>	<b>4.6</b>	<b>Bangladesh</b>	<b>11.1</b>
<b>8</b>	<b>Brazil</b>	<b>4.6</b>	<b>Japan</b>	<b>8.9</b>
<b>9</b>	<b>Italy</b>	<b>4.3</b>	<b>Philippines</b>	<b>7.8</b>
<b>10</b>	<b>Bangladesh</b>	<b>3.2</b>	<b>Egypt</b>	<b>6.7</b>

\*Table 4. is adopted from Wild S. et al.(2004) Diabetes Care 127(5):1047-1053.

It has been observed (Wild 2004) that prevalence of diabetes is similar in men and women worldwide, but there were prevalence differences by age in developing countries. The prevalence of diabetic men younger than 60 years and women at older ages was slightly higher (Wild et al. 2004). Moreover, the majority of diabetic people in developing countries worldwide were aged between 45 and 64 years, whereas the majority in developed countries was above the age of 64 years (Wild 2004).

Based on demographic changes, researchers have concluded that the “diabetes epidemics” will continue even if the obesity prevalence will remain stable until 2030, a scenario which seems unlikely. Wild estimates that the number of diabetic patients will be more than double as a consequence of worldwide population aging and urbanization.

Prospective population studies have investigated relationships between plasma glucose levels and the risk of (overall) mortality, cancer, inflammation, cardiovascular disease, and of developing diabetes (Sorkin 2005). For instance, researchers in the Baltimore Longitudinal Study found a statistically significant increase in all-cause mortality in the presence of fasting plasma glucose test results at levels  $>6.1$  mmol/l but not lower levels. For the two-hour plasma glucose test, the risk was significantly increased  $>7.8$  mmol/l (Sorkin 2005).

### **1.3 REVIEW OF THE LITERATURE: OBESITY**

One cannot dispute the negative effects of obesity. Obesity has been associated as a strong risk factor for Type II diabetes, hypertension, osteoarthritis and cardiovascular



diseases (World Health Organization 2000). In addition, poor perceived health and disability have been associated with obesity (Doll. 2000, Ford 2001).

### **1.3.1. Definitions and Classification of Obesity**

Excess adipose tissue characterizes obesity. Multiple laboratory methods have been used to quantify adipose tissue mass, including dual energy X-ray absorptiometer (DEXA), magnetic resonance imaging (MRI), and computed tomography (CT) (Lusaki 1987, Seidell et al. 1987, Gray et al. 1991, Sobol et al. 1991). The previous methods require costly equipment and have been found difficult to use in epidemiologic studies.

Body weight adjusted for stature (Body Mass Index) has been commonly used in large-scale population surveys as a surrogate for body fat content (Revicki and Israel 1986, Gray and Fujioka 1991). Body mass index (BMI), also known as Quetelet's Index, is the most commonly used tool, the ratio defined as body weight (kg) divided by height squared ( $m^2$ ). BMI has been shown to have strong correlation with body fatness, and weak correlation with height (Keys et al. 1971, Revicki and Israel 1986).

However BMI has its limitations, as it fails to distinguish between lean body mass and fat which will make the relationship between BMI and body fatness vary according to body composition (Garn et al. 1986). Also height change related to age has an influence on BMI and since the age of onset of puberty varies, estimating BMI in children and adolescents is even more difficult to determine (World Health Organization 2000). BMI provides a simple measure of obesity in adults despite these limitations.

The cut-off point for obesity has been recognized as a BMI of 30 kg/m<sup>2</sup>. The conventional accepted values (World Health Organization 2000) are shown in Table 5 (next page):

**TABLE 5.**  
**Conventional accepted values correlating Body Mass Index [BMI] and**  
**descriptions of obesity (World Health Organization 2000)**

Classification	BMI (kg/m <sup>2</sup> )	Population description
Underweight	<18.5	Thin
<b>Normal range</b>	18.5-24.9	Normal, healthy, acceptable weight
<b>Overweight</b>	≥25	
Pre-obese	25-29.9	Overweight
Obese class I	30.0-34.9	Obesity
Obese class II	35.0-39.9	Obesity
Obese class III	≥40	Morbid Obesity

\*Table 5. is adopted from The World Health Organization (2000) Obesity: Preventing and managing the global epidemic. Geneva: World Health Organization 2000 (WHO Technical Report Series 894).

No population is composed of two distinct groups, obese and non-obese. An established cut-off point of BMI 30 for obesity is completely an arbitrary one. That point only indicates greater increase in health risks above the established level, and does not mean that below BMI 30 those risks diminish (Manson et al 1995, Willet et al. 1995, 1999, World Health Organization 2000). Multiple studies have shown the difficulty to determine guidelines for healthy weight (Garn 1996, Cooper et al. 1998).

### **1.3.2.Prevalence and Trends in Obesity**

Various investigations have shown increases in prevalence of obesity in many countries including the United States (Flegal 1998), Canada (Macdonald 1997) and Australia (Eckersley 2001). Flegal in 1998 showed that in the United States, obesity prevalence, more specifically in subjects with a BMI  $\geq 35$  kg/m<sup>2</sup>, remains higher than in other countries (5 percent for white men, 10 percent for white women). Random-digit telephone surveying of 195,005 US adults aged 18 years or older (Mokdad 2001) concluded that the prevalence increase in obesity continues in both sexes, all ages, all races, all educational levels, and all smoking levels. Mokdad also concluded that obesity is strongly associated with several major health risk factors. It is worth mentioning that, in that research, BMI was calculated based on self-reported weight and height.

It has been suggested that obesity is a major avoidable contributor to the costs of illness in the U.S. (Colditz 1992). Despite those facts, the prevalence of obesity continues to grow, not in the United States alone but worldwide (Popkin Doak 1998).

### **1.3.3.Factors Associated with BMI and Obesity**

Overconsumption of food and lack of physical activity have been shown to have a causal relationship with obesity, but are not the only reasons for the obese condition. Human physiological regulatory mechanisms can be overwhelmed by many societal and environmental factors, as will be discussed.

Gender is one factor that has been associated with obesity. Multiple studies provide evidence that women have a higher prevalence of obesity, and men have a

higher prevalence of overweight, especially after the age of 50 years (Flegal et al. 1998, Stam-Moraga et al. 1999).

Several cross-sectional studies have documented increase in BMI with age (Boyle 1994, Flegal 1998). A few longitudinal studies support the previously mentioned finding, with age 60 years cited (Williamson 1990, Lewis 1997, Guo 1999).

Flegal (1998) showed variations across ethnic groups in the prevalence of obesity. Genetic predisposition for obesity might be the reason for such variations especially when individuals are exposed to an affluent lifestyle (World Health Organization 2000).

Some studies have suggested a relationship between marital status and obesity, although the relationship is not well established. Higher BMI has been associated with married subjects than subjects living alone (Kahn 1991, Rosmond 1996). Other studies suggest that no such link exists (Tavani 1994, Wamala 1997).

Studies on diet and obesity have reported inconsistent results, compromised by factors such as weaknesses in study design, methodological errors in estimating energy and nutrient intakes, and confounding factors (Lissner Heitmann 1995, Seidell 1998). Obesity has been suggested in association with a large number of various dietary factors but conclusive evidence is still lacking to prove that one diet would promote obesity more than another.

According to the World Health Organization 2000, what is commonly known as 'physical activity' actually has three components: household chores, occupational work and leisure-time physical pursuit. A large number of cross-sectional studies show an inverse association between BMI and physical activity (Rosmond 1996, Stam-Moraga

1999). Less physical activity has been observed more often in obese subjects than non-obese (Miller 1990, Cooper 2000). On the other hand, some studies have shown no association between BMI and physical activity (Seidell 1991, Tremblay et al. 1995).

#### **1.3.4. Inflammation and Obesity**

This topic will be discussed further in following sections but it remains important to introduce at this point the idea of adipose tissue and cells actively monitoring their environment. Fat cells will respond to neural, paracrine, autocrine and endocrine inputs such as prostaglandins, cytokines, steroids, cholesterol and fatty acids (Cottam 2004). Secretion of inflammatory mediators from adipose tissue in response to chronic inflammation associated with obesity was studied by Cottam et al. in 2004. Such mediators include transforming growth factor beta (TGF $\beta$ ), interleukin six (IL-6), angiotensinogen (AGT), and tumor necrosis factor alpha (TNF $\alpha$ ).

#### **1.3.5. Treatment of Obesity**

Treatment of obesity and maintaining weight loss are goals that in the literature have been proven difficult to achieve. Prevention has been shown to possibly be most effective to overcome or slow this growing epidemic (Bouchard 1996, Gill 1997). Establishing an effective strategy to prevent obesity is hampered by many challenges, including limited research and information. As suggested by James (1995), more knowledge is needed about factors contributing to obesity. Until such knowledge is obtained, changes in the prevalence of obesity and BMI over time should be observed to determine more effective strategies in both treatment and prevention.

## **1.4 REVIEW OF THE LITERATURE:**

### **ESTABLISHING THE LINKING RELATIONSHIP BETWEEN OBESITY, DIABETES AND PERIODONTAL DISEASE**

The relationship between diabetes mellitus and periodontal disease has been extensively studied by researchers, and there has been considerable interest in drawing the association between diabetes mellitus and periodontal diseases, with conflicting conclusions (Denis and Bartold, 2000). Globally both of these diseases have a relatively high incidence in the general population with a number of common pathogeneses through which periodontal infections contribute to the diabetes-related inflammatory state (Malik 2011). Researchers have found a bi-directional relationship between the two polygenic disorders with some degree of immuno-regulatory dysfunction. This means that the presence of one condition is indicated as a risk factor for the other, as well as that the meticulous treatment of either may result in improvement of the other (Malik 2011).

Chronic periodontal disease is an inflammatory condition characterized by a shift in the microbial ecology of subgingival plaque biofilms and the progressive host-mediated destruction of tooth-supporting structures (Chaffee Weston 2010). Collectively the most common diseases known to mankind, the complex classification of periodontal diseases takes into account clinical presentation, age at diagnosis, rate of disease progression, and systemic and local factors, all which may increase risk. *Gingivitis* (inflammation is confined to the gingiva, and is reversible with good oral hygiene) and

*periodontitis* (inflammation continues, and results in tissue destruction and alveolar bone resorption) (Preshaw et al. 2012) are the two current classifications of periodontal disease.

In periodontal disease, destruction of the connective tissue mostly results from the interaction of bacteria and their products with mononuclear cells (Denis Bartold 2000). The prevalence and severity of periodontal disease has been demonstrated higher among individuals with diabetes due to the accumulation of glucose mediated AGE (Advanced Glycation End-products) that would affect migration and phagocytic activity of mononuclear and polymorphnuclear phagocytic cells, leading to formation of more pathogenic sub-gingival flora (Denis and Bartold., 2000).

This inflammatory response will affect glucose-utilizing pathways by activating an infection-mediated pathway of cytokine regulation, particularly with the secretion of tumor necrosis factor (TNF- $\alpha$ ), interleukin (IL-1 $\beta$ , IL-6), and prostaglandin (PGE<sub>2</sub>) and a state of insulin resistance (Preshaw et al., 2012). The deregulated local secretion of TNF- $\alpha$  and IL-1 also mediates inflammation and the breakdown of connective tissue and alveolar bone in periodontal disease (Preshaw 2012).

In the case of diabetes mellitus (both Types I and II), the level of systemic inflammatory markers is noticeably elevated, due to the state of hyperglycemia. The elevated levels of systemic inflammatory markers contributes to both microvascular and macrovascular complications, as well as to oxidative stress and apoptosis (Dandona 2004). For instance, Engebretson and co-workers have found that TNF-  $\alpha$  serum levels in people with Type II diabetes were considerably correlated with the severity of periodontal destruction, plasma endotoxin, and IL-1b in the gingival crevicular fluid

(Engebretson 2007). Moreover, in a study of Type I diabetes, those (diabetic patients) with either gingivitis or periodontitis have higher levels of gingival crevicular fluid (GCF; fluid exudation from the gingival margins), PGE<sub>2</sub>, and IL-1 $\beta$  compared to the non-diabetic group with the same periodontal disease level (Salvi 1997).

Accordingly, it has been indicated that diabetes is a risk factor for gingivitis and periodontitis (Salvi 2008). Conversely, periodontitis may have a major role in worsening glycemic control over time among diabetic individuals and may also increase the risk of diabetic complications, by elevating levels of these cytokines closely linked to insulin resistance (Salvi 2008).

Although some previous studies have not found a significant direct effect of diabetes on periodontitis and gingivitis, many other studies have demonstrated that diabetes is a major risk factor for periodontitis (Preshaw 2012). The first clear evidence to support this hypothesis came from cross-sectional and longitudinal studies in the 1990s (Nelson) on the North American Pima Indian population (number: 2,273 subjects, age 15- 54 years). Prevalence of periodontitis among Pima Indians with Type II diabetes was 60 percent, and 36 percent in the non-diabetic patients. In a subgroup of 701 subjects (aged 15-54 years, follow up period over 2.5 years), the incidence of periodontitis was increased by approximately three-fold compared with non-diabetic Pima Indians (Nelson 1990).

Investigators have also concluded that all diabetic patients of both Type I and Type II, including children and young adults, could be at risk for development of periodontal diseases (Preshaw 2012). However the vast majority of studies were focused on the effect of Type II diabetes on periodontal diseases, possibly due to the



fact that both diseases have historically tended to appear in patients in their 40s and 50s (Preshaw 2012). One early study reported that the prevalence of attachment loss, bone loss, and gingivitis in around 10 percent of children (<18years) with Type I diabetes was greater than the control group, despite comparable plaque scores (Cianciola 1982). More recently it was found that the proportion of periodontitis in a group of 350 diabetic children (6-18 years) was greater (>20) than the 350 non-diabetic controls (8%) (Lalla 2007). In a large epidemiologic study in the United States, the risk of periodontitis among patients with poorly controlled diabetes increased 2.9-fold compared to the non-diabetic group. Conversely there was no significant increase in risk among the well-controlled diabetics (Tsai 2002).

It is likewise well-recognized that the level of glycemic control is of key importance in determining the development of chronic complications of diabetes, and may play a role in the gingival response to bacterial plaque in diabetics (Preshaw 2012). Analysis of data from the US National Health and Nutrition Examination Survey (NHANES) III identified higher significant prevalence of severe periodontitis in adults with HbA1c level >9% than those without diabetes (OR 2.90% CI 1.40, 6.03) after adjusting for the effect of confounding variables such as age, sex, ethnicity, education, and smoking (Tsai 2002). Although the metabolic control of diabetes may have an important role in the onset and progression of periodontal diseases, researchers have concluded that this association is difficult to define as it is similar to the association between glycemic control and diabetic complications such as retinopathy and nephropathy (Mealey and Oates 2006). More specifically there is marked heterogeneity in the diabetic population (Tsai 2002). The body of evidence has reported that some

poorly-controlled diabetic patients may have extensive periodontal destruction, while others may not. On the other hand, many patients with effective glycemic control may have very good periodontal health, where others may develop periodontitis (Tsai 2002).

Results from a cross-sectional analysis of Type I diabetic individuals for a mean duration greater than 16 years has demonstrated that poorly-controlled diabetics have a greater prevalence of inter-proximal attachment loss and bone loss than well-controlled Type I diabetic subjects (Mealey and Oates 2006). Corroborative longitudinal epidemiological research among the Arizona Pima Native American community found that the risk of progressive bone loss was increased 11-fold among subjects with poor glycemic control of Type II diabetes compared to a non-diabetic control group (Taylor 1998).

Conversely, not only diabetes is a risk factor for periodontitis, but periodontitis could have a negative effect that is considered a risk factor for worsening glycemic control (Preshaw 2012).

The first clear evidence supporting this hypothesis was obtained from two-year longitudinal research conducted in the Gila River Native American community (Taylor 1996). Diabetic patients with severe periodontitis at baseline had a six-fold increased risk of poor glycemic control (HbA1c > 9.0%) over diabetic patients with no periodontitis (Taylor 1996). Various investigations have reported that severe periodontitis may also increase the risk of non-oral diabetes-related complications including retinopathy, diabetic neuropathy, proteinuria, and cardiovascular complications (Preshaw 2012).

The impact of periodontitis on the development of overt nephropathy, defined as macroalbuminuria and end stage renal disease (ESRD), in patients with Type II

diabetes was investigated by Shultis as well in Gila River Native Americans (2007). Assessment of periodontal status was performed among 529 Type II diabetic patients 25 years or older, with a glomerular filtration rate of  $\geq 60 \text{ ml min}^{-1} 1.73\text{m}^{-2}$ , and without macroalbuminuria (urinary albumin: creatinine ration  $\geq 600 \text{ mg/g}$ ) (Shultis et al. 2007). Investigators found macroalbuminuria developed in 193 patients (median follow-up 9.4 years) and ESRD in 68 patients (median follow-up 14.9 years). The incidence of macroalbuminuria was 2.0, 2.1, and 2.6 times greater in patients with moderate and severe periodontitis, or in those who were edentulous, respectively, than in those with mild or without periodontitis ( $p < 0.05$ , after adjustment of age, sex, diabetes duration, BMI, and smoking), whereas the incidence of ESRD was 2.3, 3.5, and 4.9 times greater in the presence of moderate or severe periodontitis or edentulism respectively ( $p$  value  $< 0.05$ ). That research concluded that the development of overt nephropathy and ESRD in a 'dose-dependent' manner in Type II diabetes was predicted with moderate and severe periodontitis and with edentulism (Shultis 2007).

Malik et al (2011) conclude that "periodontitis may initiate or propagate insulin resistance in a manner similar to that of obesity, by enhancing activation of overall systemic immune response initiated by cytokines, and given these mechanisms promoting insulin resistance." The elevated chronic systemic inflammatory state induced by periodontal disease in people with Type II diabetes may play a major role in propagating insulin resistance and worsen glycemic control (Malik 2011). Emerging evidence indicates that periodontitis may contribute to the translocation of gram-negative oral bacteria species and their products from the periodontal biofilm into the systemic circulation, and to the translocation of cytokines from the periodontal space into the

circulation likewise. This may explain why in some studies glycemic control of diabetes showed improvement after effective periodontal therapy, which is aimed to decrease elevated serum inflammation mediators (such as TNF- $\alpha$  and IL-6) (Malik 2011).

Some researchers contend that these insights regarding effects of the treatment of periodontal infection on glycemic control of diabetes investigates only non- surgical periodontal treatment and derives from observational and treatment research in some cases statistically significant and in others not (Kiran 2005). As a treatment footnote, to date no clear evidence establishes a requirement for antibiotic use with non- surgical periodontal treatment in order to observe the improvement in glycemic control that can be associated with periodontal therapy (Kiran 2005; Chaffee and Weston 2010).

## **2.0 MATERIALS AND METHODS**

### **2.1 STUDY POPULATION**

Subjects were identified from a retrospective review of the records over a three-year period (from August 2008 to November 1, 2011) using the electronic health record (EHR) of the University of Pittsburgh School of Dental Medicine.

### **2.2 CRITERIA FOR INCLUSION**

All subjects are adult (*>age 18*) patients of record of the University of Pittsburgh School of Dental Medicine Department of Periodontics and Preventive Dentistry. Data were collected from the electronic health record (EHR) of the University of Pittsburgh School of Dental Medicine (axiUm database, Exan Systems, Las Vegas, Nevada) from the time period from August 2008 to November 2011.

### **2.3 CRITERIA FOR EXCLUSION**

Children are not included, as pediatric manifestations of periodontal disease or diabetes are not within the scope of this research. Completely edentulous [toothless] individuals would not be reflected in the electronic health record, as periodontal disease is not present in conjunction with full edentulism. Subjects physically, mentally, or legally incapacitated so that informed consent to treat cannot be obtained are not included.

## **2.4 PERIODONTAL ASSESSMENT**

*Pocket depth upon probing* is the measure from the free gingival margin (FGM) to the base of the sulcus/pocket with the use of a calibrated probe (*HuFriedy Michigan 0 probe, diameter of probe is 0.5 mm*). Pocket depth is probed on six sites per tooth (distobuccal, midbuccal, mesiobuccal, distolingual, midlingual, and mesiolingual).

Clinical periodontal attachment level and the loss of periodontal attachment are defined as the distance in millimeters (mm) from the cementoenamel junction (CEJ) to the base of the pocket. Probing depth is the distance from the free gingival margin (FGM) to the base of the sulcus/pocket.

## **2.5 SPECIFIC DATA ELEMENTS EXTRACTED FROM THE ELECTRONIC HEALTH RECORD**

- Height and weight to compute BMI.
- Patient's diabetic status, as reported in the EHR.
- Smoking status.
- Race.

## **2.6 STUDY DESIGN**

Subjects used in this study were 7171 dentate patients (n=7171), mean age 54 years. Patients' records with documented weight, height, age, sex, Type II diabetes status (self-reported) and a completed periodontal evaluation were included. Periodontal disease was classified in the presence of periodontal pocketing of 4mm or greater (any

surface or tooth). Body mass index (BMI) was calculated and patients were classified as obese ( $BMI \geq 30$ ) or non-obese ( $BMI < 30$ ).

## 2.7 STATISTICAL ANALYSIS

In this study, 7,171 records were reviewed, 5751 white or Caucasian to 1420 African-American ethnicity. Among the total dental treatment records, twice as many, or specifically 5104, had recorded periodontal disease versus 2067 medical records that did not record periodontal disease. In the total set of electronic health records reviewed, 5263 did **not** calculate BMI to the level of obesity, while in 1908 of the EHR, height and weight calculations fell into the category of calculated *obesity*.

The EHR population sample was split nearly 50:50 male to female, 3351 males to 3820 females, the relationship of gender to periodontal disease was statistically significant,  $p = 0.038$ . Similar to the calculated obesity, the total EHR were dominantly non-smokers (4364) versus 2807 individuals who admitted cigarette smoking or other tobacco use. The relationship of smoking to periodontal disease was not statistically significant  $p = 0.089$ , and the relationship was not used in the final model.

Step-wise logistic regression analysis was performed, the final model including the variables BMI, age, obesity, sex, and race. Relationships between periodontal disease and those variables were significant, BMI  $p = .000$ ; age  $p = .000$ ; sex  $p = .038$ ; obesity  $p = .037$ ; and African-American ethnicity  $p = .012$ . (Please see the tables for more detail.)

The results showed a strong relationship between periodontal disease and obesity. There was also a strong relationship between periodontal disease, age, and ethnicity. Statistical analysis of the retrospective data collected established positive association between periodontal disease and obesity ( $p = 0.000$ ).

### 2.7.1 Tables Illustrating the Statistical Analysis

**TABLE 2.7.1.1**  
**Frequency table. Periodontal involvement : Obesity : Diabetes Mellitus**

	Frequency	Percentage	Cumulative Percentage
<b>Periodontal involvement</b>			
<b>Absent</b>	2067	28.8	28.8
<b>Present</b>	5104	71.2	100.0
<b>Obesity</b>			
<b>Absent</b>	5263	73.4	73.4
<b>Present</b>	1908	26.6	100.0
<b>Diabetes mellitus</b>			
<b>Absent</b>	6488	90.5	90.5
<b>Present</b>	683	9.5	100.0



**TABLE 2.7.1.2**  
**Frequency table. Sex : Smoking : Race/Ethnicity**

	Frequency	Percentage	Cumulative Percentage
<b>Sex</b>			
Female	3820	53.3	53.3
Male	3351	46.7	100.0
<b>Smoking</b>			
Absent	4364	60.9	60.9
Present	2807	39.1	100.0
<b>African American</b>			
False	5751	80.2	80.2
True	1420	19.8	100.0

**TABLE 2.7.1.3**  
**Model Summary**

**Model Summary**

Step	-2 Log likelihood	Cox & Snell R Square	Nagelkerke R Square
1	3748.868 <sup>a</sup>	.101	.216
2	3750.601 <sup>a</sup>	.101	.215

a. Estimation terminated at iteration number 6 because parameter estimates changed by less than .001.

**TABLE 2.7.1.4**  
**Classification Table**

Classification Table<sup>a</sup>

Observed			Predicted		
			DM		Percentage Correct
			Absent	Present	
Step 1	DM	Absent	6452	36	99.4
		Present	656	27	4.0
	Overall Percentage				90.4
Step 2	DM	Absent	6448	40	99.4
		Present	657	26	3.8
	Overall Percentage				90.3

a. The cut value is .500

**TABLE 2.7.1.5**  
**Variables in the equation**

Variables in the Equation

		B	S.E.	Wald	df	Sig.	Exp(B)
Step 1 <sup>a</sup>	BMI	.085	.009	94.838	1	.000	1.089
	Age	.047	.003	293.221	1	.000	1.048
	Sex(1)	-.175	.088	3.968	1	.046	.839
	Periodontitis(1)	-.313	.115	7.334	1	.007	.731
	Obese(1)	-.272	.132	4.242	1	.039	.762
	Smoking(1)	-.117	.089	1.739	1	.187	.889
	AfricanAmerican(1)	-.249	.101	6.078	1	.014	.780
	Constant	-6.542	.377	300.714	1	.000	.001
Step 2 <sup>a</sup>	BMI	.085	.009	94.150	1	.000	1.089
	Age	.047	.003	294.226	1	.000	1.048
	Sex(1)	-.182	.088	4.316	1	.038	.833
	Periodontitis(1)	-.324	.115	7.932	1	.005	.723
	Obese(1)	-.276	.132	4.372	1	.037	.759
	AfricanAmerican(1)	-.255	.101	6.385	1	.012	.775
	Constant	-6.563	.377	303.771	1	.000	.001

a. Variable(s) entered on step 1: BMI, Age, Sex, Periodontitis, Obese, Smoking, AfricanAmerican.

### **3.0 DISCUSSION**

The relationship between diabetes and periodontal disease has been well documented and may be two-way (Grossi 1998). Strong evidence establishes that diabetes is a risk factor for gingivitis and periodontitis (Papapanou 1996; Mealey 2003). Landmark research by Löe (1993) determines that severe periodontal disease is the sixth major complication of diabetes.

Multiple studies have also related periodontal disease to obesity (Saito 1998, Al-Zahrani 2003). Genco and Grossi in 2000 and 2005 have clearly related obesity to periodontal disease through the pathway of insulin resistance.

The results of this retrospective research reveal that a statistically significant relationship did exist between obesity and periodontal disease. These results compare favorably with the systematic meta-analysis of results from 57 independent populations performed by Chaffee and Weston in 2010.

Results in the present study have also shown a positive association between periodontal disease and diabetes mellitus. Similar results have been shown elsewhere. Periodontal disease relative to diabetes is the best example of the clinical recognition of periodontal inflammation as an indicator of a potential underlying systemic condition (Cianciola 1982; Genco 1990; Pucher 2004). Other studies have shown only marginal support to the relationship between glycemic control and the extent or severity of periodontitis (Bridges 1996), whereas some studies found no evidence, or failed to

reach statistical significance establishing such a relationship, between glycemic control and periodontal status (Barnett 1984; Sastrowijoto 1989).

The concept that insulin resistance appears to mediate the relationship between obesity and periodontal disease, put forth by Genco and Grossi 2005, is supported by the findings. Results show a statistically significant relationship between obese patients with periodontal disease and a higher risk for diabetes mellitus. In obesity as in periodontal disease, there is a chronic stimulation and secretion of pro-inflammatory cytokines which could contribute to insulin resistance (Nishimura et al 2001).

For this study, smoking (pack years), age, sex and race were controlled and the parameters again analyzed using multiple regression analysis. Results again showed a statistically significant relationship between the studied parameters. Not only do confounding variables make analysis difficult to interpret, but also the fact that some variables were self-reported may introduce an inherent bias in the study.

This study investigated the association between obesity and periodontal disease, and also the association of periodontal disease with diabetes. Specifically this research attempted to determine whether obese individuals (assessed by variable BMI) are at greater risk of developing periodontal disease (assessed by mean clinical attachment loss), and whether obese individuals with periodontal disease are at greater risk for diabetes mellitus (measured as poor glucose control). Electronic health records (n=7171) obtained from the University of Pittsburgh School of Dental Medicine axiUm database were analyzed. Review of the literature regarding periodontal disease, obesity, diabetes mellitus and the relationships linking all three conditions together was made. Due to the variability and complexity of the associated parameters, further

investigation is necessary to elicit and clarify whether such association exists beyond reasonable doubt.

## 4.0 CONCLUSION

The records of 7171 patients aggregated from the electronic health record (EHR) at the University of Pittsburgh School of Dental Medicine were reviewed and analyzed to ascertain whether a correlation existed between obesity and periodontal disease. When BMI values were compared to attachment loss a statistically significant relationship was found ( $p=0.000$ ). When the confounding variables of smoking, age, race, sex and diabetic status were controlled for, a statistically significant relationship remained between increasing body mass index and attachment loss ( $p=0.000$ ).

Due to accumulating evidence in research linking periodontal disease to multiple systemic conditions, the dental health-care provider must be cognizant of the periodontal status of patients showing indication of such systemic conditions associated with chronic inflammation (most specifically in this regard, diabetes and metabolic syndrome). Numerous studies have identified obesity as a major public health concern (Mokdad 1999) and the global incidence and prevalence of obesity has been on the rise worldwide.

Further understanding of how obesity is linked to periodontal disease, along with more well-designed prospective studies, is necessary to clarify how such relationships between periodontal health and systemic health exist. Prevention is key for both chronic conditions. Health-care providers should continue to underscore for their patients the importance of reaching and maintaining a healthy weight, following the dietary and nutritional recommendations of the U.S. National Institutes of Health, and having regular oral health examinations and screenings for signs of periodontal disease.

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